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Letter to the Editor: Brief Case Report

Cotard's Syndrome as a Neuropsychiatric Sequela of COVID-19



Key words: COVID-19, Cotard's Syndrome, delirium.

TO THE EDITOR Since the Coronavirus pandemic (COVID-19) began in December 2019, there have been over 167 million cases and 3.5 million deaths.¹ While the pandemic is entering a new vaccination phase, the neuropsychiatric sequelae continue to cause distress in those with COVID-19. Cotard's syndrome (CS) is a rare constellation of symptoms described by Jules Cotard in 1880. Cotard described it as anxious melancholia, suicidal behavior, insensitivity to pain, delusions of nonexistence, and delusions of immortality.² The full syndrome has gone through multiple versions of symptomatic framework and was first described as encompassing more than the memorable delusion of negation. Currently, the delusions of nonexistence and immortality are central to the syndrome and, although counterintuitive, can exist together. The epidemiology and pathophysiology are not clearly documented, but the syndrome has been described in a wide range of neuropsychiatric illnesses including schizophrenia, mood disorders, neurosyphilis, subdural hemorrhage, brain neoplasms, migraines, neurocognitive disorders, medication toxicity, traumatic brain

injury, and encephalitis.³ It has been described in the context of delirium in one case report,⁴ but not in the context of delirium from underlying COVID-19 described in the following case.

Ms. G, a 52-year-old female with no past psychiatric history and a history of type 2 diabetes mellitus, obstructive sleep apnea, and congestive heart failure, presented to the emergency department with fever, respiratory failure, agitation, and thoughts that her son was dead. She was hospitalized for acute on chronic respiratory failure and delirium. This admission was five days after treatment for COVID-19 pneumonia with remdesivir, dexamethasone, and a new oxygen requirement of 3 liters per minute. Initial psychiatric examination was consistent with delirium with inability to maintain attention, disturbances in perception, and distressing thoughts that her son was dead. A Mini-Mental State Exam could not be completed at admission because of severe agitation. On hospital day (HD) 1–5, she was administered haloperidol 5 mg oral three times a day for severe agitation, hallucinations, and delusions secondary to delirium. On HD 6, she described the belief that she was dead and that no one could hear her. At this time, her Mini-Mental State Exam was 16/30 with poor construction, memory, attention, and orientation. On HD 7, despite her Mini-Mental State Exam improving to 28/30, she described new delusions. She said that her hand was dead and “black” from blood loss. She reported her mood as “sad” throughout her hospital stay until delirium resolved. Neurologic

examination was nonfocal and symmetric. CT head without contrast showed an unchanged small focus of encephalomalacia within the left superior frontal gyrus. Haloperidol was tapered and discontinued on HD 9, and she was discharged home on HD 10 with resolution of symptoms, although still distressed that she had believed her son was dead.

This case report expands the current literature, showing that CS occurs in patients after acute COVID-19 and adds to the differential diagnosis of known neuropsychiatric sequelae of COVID-19. This was a patient with no past psychiatric history or focal neurologic findings, presenting with CS in the context of delirium and COVID-19. This is the second report of CS in delirium. The first case report described a 76-year-old female with no past neurologic or psychiatric illness who developed delirium after a fall, acute kidney injury, rhabdomyolysis, and acute liver failure. She believed that she was missing 2 milliliters of blood, that she was dead, and that she was also immortal. She improved on antibiotics and quetiapine 50 mg daily.³ While there is no defined neurobiology underlying CS, Tomasetti et al. summarized the current literature, which suggests a hypoactive default mode network with a hyperactive basal ganglia and thalamus, leading to the inability to process the idea of self and to the exaggeration of emotional and somatosensory stimuli.⁵ This patient might have been more susceptible to the development of delusions in the context of COVID-19 because of the encephalomalacia in her left superior frontal gyrus, as this area is potentially

linked to the default mode network.⁶ With other case reports describing psychosis secondary to COVID-19, this case report emphasizes the importance of increasing COVID screening in patients with new psychiatric symptoms and also the importance of thorough psychiatric screening for patients with COVID-19.

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Human Subject Research Statement: This case report was approved by the IRB as exempt.

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